AMENDMENT

Kindly amend the specification, without prejudice, without admission, without surrender of subject matter, and without any intention of creating any estoppel as to equivalents, as follows.

IN THE CLAIMS:

Kindly amend the claims, without prejudice, without admission, without surrender of subject matter, and without any intention of creating any estoppel as to equivalents, to read as follows:

1. (Previously presented) A transgenic mouse model showing hypomyelinosis of the thalamus wherein the transgenic mouse comprises a homozygous disruption in chromosomal DAP12 (DNAX Activation Protein 12) gene function, and wherein the homozygous disruption includes the promoter region and exons 1, 2, and 3.

2. (Canceled)

3. (Currently amended) The transgenic mouse model of claim 1, wherein the homozygous disruption in DAP12 can be pheonotypically exhibited as a myelinogenesis developmental disorder or a neuropsychiatric disorder associated with disruption in DAP12 gene function.

4.(Currently amended) The transgenic mouse model of claim 3, wherein the neuropsychiatric disorder is selected from the group consisting of Nasu-Hakola disease, dementia associated with disruption in DAP12 gene function, schizophrenia associated with disruption in DAP12 gene function, schizotypal personality disorders associated with disruption in DAP12 gene function, obsessive-compulsive disorders associated with disruption in DAP12 gene function, or Tourette's syndrome associated with disruption in DAP12 gene function.

5. (Currently amended) The transgenic mouse model of claim 3, wherein the neuropsychiatric disorder is Nasu-Hakola disease or dementia associated with disruption in DAP12 gene function.

6-18. (Canceled)

19. (Previously presented) The transgenic mouse model of claim 1, wherein the expression of myelin basic protein in the brain is weak in regions where DAP12 is strongly expressed in wild-type mice.

20. (Previously presented) The transgenic mouse model of claim 1, wherein the transgenic mouse exhibits an impairment in sensorimotor gating as compared to wild-type mice.